Understanding the 12-lead ECG, part II

**Bundle-branch blocks**
- Most common electrocardiogram (ECG) abnormality
- Appears as a wider than normal QRS complex
- Occurs when one of the two bundle branches can’t conduct the impulse
- Most common cause: ischemic heart disease

**Right bundle-branch block (RBBB)**
- Impulse conduction to right ventricle is blocked
- Examine lead V₁ to identify RBBB
- ECG show delayed or positive R wave
- Key identifier is QRS complex wider than 0.12 second, with positive R wave in V₁

**Left bundle branch block (LBBB)**
- Electrical impulses don’t reach left side of the heart
- QRS wider than 0.12 second
- Key to recognizing LBBB is a wide downward S wave or rS wave in leads V₁ and V₂

**First Degree AV Block**
- Delay in electrical conduction from SA node to AV node
- Electrical stimulus is prevented from traveling to ventricular conduction system

**First Degree AV Block - Criteria**
- Rhythm - regularity between P-P interval and R-R interval is constant.
- Rate - normal, 60 - 100 beats per minute
- P wave configuration
  - Same configuration and shape
  - P wave occurs before each QRS complex
Second Degree AV Block, Mobitz I (Wenckebach)

- Some electrical impulses blocked/non-conducted from SA node to ventricles at AV junction region.
- AV node conducts electrical impulse to ventricular conduction pathway until it fails, then resets in a repetitious pattern.

Second Degree AV Block, Mobitz I (Wenckebach) - Criteria

- Rhythm
  - R-R interval regular
  - RR interval irregular due to blocked impulses
- Rate
  - Atrial rate within normal limits
  - Ventricular rate slower than atrial rate
- P wave configuration
  - Normal size
  - Upright
  - One P wave for every QRS complex, with additional P waves
- PR interval - varies, starts short, gets progressively longer until QRS wave is dropped, then cycle is restarted
- QRS duration - normal .06 - .10 seconds

Second Degree AV Block, Mobitz II

- P wave configuration
  - Normal size
  - Upright
  - One P wave for every QRS complex, but additional P waves
- PR interval - constant, even after QRS drop occurs
- QRS duration - normal, .06 - .10 seconds
What do you think?

• Sinoatrial block, type II
• Second-degree atrioventricular (AV) block, type I
• Second-degree AV block, type II
• Nonconducted atrial premature impulse

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• Sinoatrial block, type II
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What do you think?

• Second-degree AV block, type II

P waves occur regularly in this tracing; some of them are conducted to the ventricles while others are blocked; therefore, it is second-degree AV block. In this tracing, when the P waves are conducted, the PR intervals do not lengthen; therefore, this is second-degree AV block, type II.

Third Degree AV Block

• AKA complete heart block (CHB)
• All electrical impulses originating above the ventricles are blocked.
• No correlation exists between atria and ventricle depolarization.

Third Degree AV Block - Criteria

- Rhythm
  - P-P interval is regular.
  - R-R interval is regular, but different from P-P interval.
- Rate
  - Atrial rate is 60 - 100 beats per minute.
  - Ventricular is 20 - 40 beats per minute.
- pauses in the middle of a regular rhythm.
- there are no extra P waves during the pauses -- an indication that this is not AV block.
- the pause is exactly twice the length of the shorter cycle, indicating regularly firing sinus impulses that fail to conduct to the atrium at times;

This is SA block. Because the pause is twice the shorter cycle, it is type II.

Recognizing myocardial infarction (MI)
- Series of predictable ECG changes occur in MI
- ST-segment-elevation MI (STEMI) - serious type of MI, associated with more complications, higher risk of death

Characteristic changes in AMI
- ST segment elevation over area of damage
  - ST depression in leads opposite infarction
  - Pathological Q waves
  - Reduced R waves
  - Inverted T waves

ST elevation
- Occurs in the early stages
- Occurs in the leads facing the infarction
- Slight ST elevation may be normal in V1 or V2

Deep Q wave
- Only diagnostic change of myocardial infarction
- At least 0.04 seconds in duration
- Depth of more than 25% of ensuing R wave

T wave changes
- Late change
- Occurs as ST elevation is returning to normal
- Apparent in many leads
**Bundle branch block**

- Anterior wall MI
- Left bundle branch block

**Sequence of changes in evolving AMI**

- 1 minute after onset
- 1 hour or so after onset
- A few hours after onset
- A day or so after onset
- Later changes
- A few months after AMI

**Inferior wall STEMI**

- Elevated ST segments in leads II, III, and aVF, which monitor the heart’s inferior or bottom wall
- Area of the heart perfused by the right coronary artery

**Inferior infarction**

- Directly to the left of the septal area
- Also perfused by the LAD
- Most muscular, powerful pumping wall of the heart, responsible for large proportion of cardiac output
- ST elevation seen in V3 and V4

**Septal MI**

- Perfused by the left anterior descending (LAD) coronary artery
- ST-segment elevation seen in leads V1 and V2, the precordial or chest leads located on the anterior chest wall over the septum

**Anterior-wall STEMI**

- Directly to the left of the septal area
- Also perfused by the LAD
- Most muscular, powerful pumping wall of the heart, responsible for large proportion of cardiac output
- ST elevation seen in V3 and V4
Anterior infarction

- Perfused by the circumflex artery
- Muscular, contributes significantly to the heart’s pumping ability
- Monitored by precordial (chest) and frontal (limb) leads
- ST-segment elevation will appear in leads I, aVL, V5, V6

Lateral infarction

- Monitored by precordial (chest) and frontal (limb) leads
- ST-segment elevation will appear in leads I, aVL, V5, V6

Common dysrhythmias

- Sinus bradycardia
  - Sinus rhythm slower than 60 beats per minute
  - Commonly caused by ischemic heart disease causing sinoatrial (SA) node to malfunction
  - Also seen in MI, some medications (such as beta-blockers), and well-conditioned athletes

Sinus bradycardia

- Signs and symptoms: hypotension, lethargy, fatigue, chest pain, difficulty breathing
**Sinus tachycardia**
- Sinus rhythm faster than 100 beats per minute
- Related to physiologic cause: fever, infection, pain, physical exertion, anxiety, shock, hypoxia
- May need beta-blocker if cause unknown

**Atrial fibrillation (AF)**
- Common dysrhythmia
- Irregular heart rhythm with no meaningful P waves
- Atrial kick lost, atrias quiver due to depolarization of atrial cells
- Causes irregular ventricular rate, 40 to 180 beats per minute

**Premature ventricular contractions (PVCs)**
- Wide abnormal premature QRS complex
- Due to conduction through the ventricle instead of His-Purkinje system
- QRS greater than 0.12 second
**Ventricular Tachycardia (VT)**
- Rapid rate, 100 to 250 beats per minute
- Wide, bizarre, QRS complex followed by large T wave
- Patient may be unconscious, pulseless, apneic—initiate CPR
- If patient awake, treat as medical emergency

**Ventricular Tachycardia (Vtach)** - Criteria
- Rhythm
  - P-P interval usually not identifiable.
  - R-R interval usually regular, can be slightly irregular, at times.
- Rate
  - Atrial rate cannot be determined.
  - Ventricular rate - 100 - 200 beats per minute.

**Ventricular Fibrillation (Vfib)**
- Chaotic asynchronous electrical activity within ventricular tissue results in no cardiac output.

**Ventricular Fibrillation (Vfib) - Criteria**
- Rhythm
  - P-P interval cannot be determined.
  - R-R interval, if able to determine, will be irregular.
- Rate
  - Atrial rate cannot be determined.
  - Ventricular rate, if identifiable, will be greater than 300 beats per minute.
Ventricular Fibrillation (Vfib) - Criteria

- P wave configurations - not identifiable
- PR interval - cannot be identified
- QRS duration - cannot be determined

Asystole

- AKA straight line or flat line
- No electrical activity is present in the myocardium.

Asystole - Criteria

- Rhythm - no P-P or R-R intervals are present.
- Rate - no atrial or ventricular rates are present.
- P wave configurations - no P waves are present.
- PR interval - none, since no waveforms are present.
- QRS duration - not measurable, no waveforms are present.

Electrolyte Disturbances

- Hyperkalemia
  - Peaked T waves
  - PR prolongation and P wave flattening
  - QRS widening
- Hypokalemia – ST depression, T wave flattening, U waves
- Hypocalcemia – Prolonged QT interval
- Hypercalcemia – Shortened QT interval

Drug and Electrolyte ECG Effects
Hyperkalemia
- Peaked T waves
- PR prolongation and P wave flattening
- QRS widening

Hyperkalemia

Hypokalemia
- ST depression, T wave flattening, U waves
- Prolonged QT interval

Hypocalcemia
- May be due to pancreatitis or kidney failure
- Prolonged QT interval

Hypercalcemia
- Shortened QT interval (short/absent ST segment)

Digitalis
- Cardiac glycoside used to either
  - Treat congestive heart failure (positive inotropic effect)
  - Treat atrial fibrillation or flutter
  - Effects myocardium (arrhythmias) by shortening repolarization
  - Makes ST and T “fuse” together, creating a scooping effect of the ST segment

Digitalis
- Scooping of ST segment
- Shortening of QT interval
- Low amplitude of T wave
- Elongation of PR interval
- High amplitude of U wave
Atrial tachycardia with AV block

AF with accelerated junctional rhythm

Mobitz I

Digitalis poisoning

1st degree AV Block

Bidirectional VT

Ventricular bigeminy

Digitalis poisoning

Bidirectional VT

Ventricular bigeminy

Digitalis poisoning

1st degree AV Block

Quinidine

- Anti-arrhythmic
- Prolongation of QT interval and U

Quinidine effects and early toxicity

Tricyclic antidepressants (TAD)

Sinus tachycardia

QRS > 100 msec

Rv1 > 3 mm

Sinus tachycardia with a prolonged QRS interval

Rightward axis

Tall R wave in lead aVR

Markedly abnormal repolarization changes suggests TAD poisoning

HYPOTHERMIA

Sinus bradycardia with first-degree AV block is evident.

The downstroke of each QRS complex is slurred and is typical of a J (Osborne) wave.